Periscope.

PATHOLOGICAL.

Contribution to the Study of Syphilis of the Nervous System.—F. Raymond (*Archives de Neurologie*, No. 83, 1894) records three cases of interest, and gives anatomical and pathological details of value. The first case showed diffuse vascular lesions of the cerebral cortex, the direct continuation of gummatous lesions of the large vessels; the second likewise presented vascular lesions, but they were limited to the cortex; in the third there was a similar irritative process, but more circumscribed in its vascular areas, and existing in the smallest capillaries only. In these three cases, the cerebral lesion was also associated with diffuse lesions of the spinal cord, evidently syphilitic. The first two cases were syphilitics, who became paretic. Raymond looks upon general paresis of syphilitic origin as a diffuse vascular encephalitis. But all subjects of diffuse vascular encephalitis are not syphilitic. General paresis supervenes only when nervous elements begin to suffer from defects in the apparatus of nutrition. The period of resistance may be very long and symptoms not well marked in consequence. But at a certain moment the nerve-tissue gives way; and the encephalitis, up to this point purely vascular, becomes mixed: elements of the neurolgia hypertrophy become sclerosed, nerve tubes are destroyed, beginning with the superficial ones of the convolutions, nerve cells undergo a change, and the injury to the cortex is now permanent and irremediable. Beside the specific agent, heredity must also be considered as a factor in the weakness or stability of nerve elements Certain syphilitics can stand an interstitial encephalitis for a long time without becoming paretic; they may even recover if nerve tissue remains intact. Others, again, fall quickly into dementia, because their nervous structure possesses no power of resistance. BRYSON.

Pathological Anatomy of Multiple Sclerosis.—Dr. E. W. Taylor, of Boston (*Deutsche Zeitschrift für Nervenheilkunde*, V. B. I). The paper comprises a report of three cases of multiple sclerosis of ordinary clinical course, with careful microscopical examination.

The points of special interest in the various cases are: (*a*) The unusual extent of the pathological process at several levels, involving a complete transverse degeneration of the cord. (*b*) The equal affection of gray and white matter. (*c*) The degeneration of anterior and posterior roots. (*d*) The involvement of the nuclei of all the cranial nerves in medulla pons and region of corpora quadrigemnia. (*e*) A patch of continuous sclerosis, implicating the central canal of lower medulla. The entire floor of fourth ventricle, and the aqueduct of Sylvius, so far as the nucleus of the third nerve. (*f*) The degeneration of fibres of the cauda equina. (*g*) The involvement of the cerebral and cerebellar cortex and the inner capsule.

The general conditions to be drawn from the examination are, that contrary to the popular opinion, white and gray matter are equally attacked; that there is no point of predilection in the central nervous system for the appearance of the sclerotic patches; that the cortex of both cerebrum and cerebellum does not escape the degenerative process.

The presence of lesions in all cranial nerves is a matter of interest in view of previous opinion on that point. The degeneration of fibres of the cauda equina is of importance as throwing light on the possible extension of the process to the peripheral system. The sharply defined edges of many of the patches studied is worthy of mention, an observation contrary to Charcot's original description.

Secondary degenerations are not observed. Ganglion cells are in many cases entirely normal; in others, where the process is in a more advanced stage, varying conditions of degeneration, characterized especially by a much greater degree of pigmentation than normally occurs. Blood vessels, though in places somewhat affected, do not appear to stand in causal relation to the formation of the sclerosis, for the following reasons: (1) The patches are not always associated with diseased vessels. (2) The vessels, even in much degenerated areas, often show no marked changes. (3) Disease of vessels, in one case examined, was enterely lacking. COLLINS.

The Pathological Anatomy of Cerebro-Spinal Meningitis. Dr. G. Bikeles (*Centralbl. f. Nervenkr. u. Psych.*, Aug., 1894) caused light epileptic seizures in guinea pigs by tapping them on the head with a percussion hammer (Westphal's method). Afterwards these animals showed symptoms of motor disturbances and muscular paresis, which in one case were accompanied by tonic and clonic contractions. Several weeks later the animals were killed. The brain and spinal cord were stained by Marchi's method (Muller's fluid and osmic acid), and examined microscopically. Throughout the entire central nervous system degeneration of the medullary fasciculi and sheaths were found. There were no hæmorrhages. Characteristic of all the cases was the fact that the degeneration did not limit itself to the seat of the injury. From these experiments it would appear that cerebral concussion causes a destruction of the medullary fasciculi in the entire central nervous system. The process mav be considered as a traumatic degenerative neuritis, inasmuch as an injury may not only cause swelling of the axis cylinders, but also destruction of the medullary sheaths. MACALESTER.

Changes in the Arteries at the Base of the Brain in the Sane and Insane.—Mori was led to examine carefully the brains of seventy bodies, of which thirty-five died free from any mental trouble, and thirty-five were insane.

He found that of the sane thirteen presented anomalies of the cerebral arteries, nine of the cerebellar, and thirteen showed no variation.

Of the insane, thirty-two presented anomalies of the cerebral, one of the cerebellar arteries, and two were normal.

The anomalies in the cerebral arteries of the sane consisted in difference of calibre of the posterior communicans. In ten of the insane the posterior cerebral was given off from the internal carotid; in five the posterior communicans was absent; in nine the anterior communicans was duplicated; and in five there was found an accessory artery of the corpus callosum.—*Monitor Zoologico Italiana*, Anno 40, No. 10. KRAUSS.

Degeneration and its Stigmata.—C. L. Dana, M. D. (*Med. Rec.*, Dec. 15, 1894). The final work of modern physiognomy will not be to fix upon human beings any stigma which marks them as necessarily useless, defective or dangerous. We have not discovered a type of criminal man, or of the insane man, or epileptic or neurotic man. All these have common marks, which show simply that they belong to the same somewhat handicapped family. Their presence in an individual will show that he must be especially careful in educating and using his natural power. The discovery of degeneracy throws an additional responsibility upon him, for there are few so bad but that, with a proper environment, they can get along successfully in life. There may be some born criminals, but they are very few; most are simply persons of degenerate type who fail to husband properly the endowments they possess. We do not excuse the cripple who attempts to become a sprinter, nor should we excuse the morally defective who indulges in debasing habits and low temptations. All modern studies seem to show that man must be more than ever careful of his education, his training, and his surroundings, and of using all possible moral and spiritual agencies to strengthen his defects and make his powers more stable. The future of the degenerate depends enormously upon these factors, and his responsibility lies in his following that line of life which is right for him. By this means one can render many defects harmless, and make the unstable almost as firm in judgment and healthful in body as the happy possessors of perfectly balanced organizations.

FREEMAN.

The Pathological Anatomy of Chorea Chronica Progressiva Hereditaria.—Oppenheim and Hoppe (*Arch. f. Psych. u. Nervenkr.*, Bd. xxv., Hft. 3). observed two cases of this disease. One was a woman, fifty-six years old, who had been afflicted for sixteen years. The other was a man, seventy-five years old, who had suffered for five years. In both cases a hereditary predisposition was traceable Autopsy showed the viscera to be normal. The brains presented, microscopically, considerable changes: narrowing of the convolutions in the motor regions, and of the pareital and occipital lobes; also, widening of the sulci. In the subcortical regions of these parts, small areas of fresh hæmorrhagic imflammation, and older ones with a fibrillary structure, were found on microscopic examination. There were also similar conditions in the pons and medulla. The pyramidal cells were normal, but the small round cells between the first and second cortical layer were diminished in number, especially in the motor region. In the cervical enlargement of the cord there was proliferation and swelling of the glia cells, and thickening of the meshes and blood vessels. The peripheral nerves showed distinct degeneration. The authors consider the process to be a disseminated cortical and subcortical encephalitis. The cortical atrophy is the consequence of the encephalitis. In the first case there developed hydrocephalus externus subsequently to the cortical atrophy. The changes in the cord are not to be considered secondary; they are caused by the conditions of the blood vessels, and are not systemic. The changes of the nerve cells are doubtful because the influence of *la grippe*, of which the first case died, and the old age of the second cannot be excluded.

MACALESTER.

Histological Alterations in the Cerrbral Cortex in Some Mental Disease.—Colella (*Archives Italiennes de Biologie*, 1894, p. 216). Careful examination of the cortex of the brain in a case of progressive general paralysis with syphilis, in paralysis with alcoholism, and one of alcoholism, by the Gold method, has led the author to the following conclusions:

In general paralysis preceded by syphilitic infection, the histological changes are confined mainly to the blood vessels, to the neurolgia cells, as well as to the cell protoplasm and the protoplasmic branches. The axis cylinder prolongations are but rarely affected, and when this does occur it is rather late in the disease. Pathological alterations show themselves first in the blood vessels.

In dementia paralytica associated with or due to alcohol, there is found hypertrophy of the spider cells in conjunction with different stages of nutritional disturbances in the axis cylinder prolongations, with but rudimentary changes in the protoplasmic prolongations. In these cases the blood vessels do not show signs of degeneration.

In chronic alcoholismus the changes are overwhelmingly parenchymatous in their manifestation. The axis cylinder prolongations are involved, and certain parts of the cell body and the protoplasmic ramifications. The blood vessels and neurolgia remain unaffected.

Finally, the writer concludes that the protoplasmic prolongations and the nerve prolongations have a different physiological significance, and especially have the protoplasmic prolongations a close relationship to neurolgia cells and blood vessels, so that they must play an important role in the nutrition of the nerve tissues. COLLINS.

A New Theory of the Causes of Some Nervous Diseases, Especially of Neuritis and Tabes.—Edinger.

The theory is based upon the following laws:

1. The functionation of every organ always causes molecular changes to take place in it; the organ suffers a certain damage which must be repaired. When this restitution is adequate to the amount of function, the organ is strengthened by functionating; in the other case it undergoes retrogressive changes.

2. A damaged or weakened tissue soon decays and makes place for the surrounding tissues which grow into it. What has heretofore been called proliferation, hypertrophy, interstitial inflammation, etc., has been proven by Weigert to be only an ingrowth of healthy tissue into diseased tissues.

In the so-called gray atrophies of the central nervous system the cardinal tissue is always first diseased, after which this weakened tissue becomes proliferated and retrograde; then destroyed by the neurolgia. This overgrowth of the neighboring tissues cannot but damage the whole organ. This overgrowth of interstitial tissue, caused by decay of the motor fibres in a nerve, must also damage the sensory fibres contained in it.

Edinger thinks that by appreciating the importance of these facts, many observations, heretofore unexplained, can be comprehended.

If there is a disproportion between function and restitution in such a manner that the restitution is not sufficient to cause the tissue to return to its former condition, a degeneration of the tissue must follow. This disproportion will occur in case of absolute overfunction—when, for instance, a certain group of muscles or nerves is over-exerted. In some cases, even where the restitution would be complete for a normal amount of function, it would occur still easier if, aside from over-function, restitution was impairedby deficient nutrition (as Edinger finds it to be the case in the so-called occupation paralysis of cigar rollers, etc.). But even with a normal amount of function nutrition may be so deficient as to prevent sufficient restitution. In the latter case those parts which perform comparatively the largest amount of function would have the greatest disposition to degenerate.

That there are diseases of the spinal cord which are intimately connected with disturbances of nutrition in the central organ, is proven by the existence of tabic symptoms in diabetes, by degenerations of the posterior columns or pyramidal tracts of dogs in whom artificial circulatory disturbances are produced by rapid rotation, etc.

It will also be easier to comprehend why multiple neuritis is seen accompanying or following such a large number of diseases (anæmia, tuberculosis, contagious disease, as measles, scarlet fever, etc.), by assuming that as a consequence of exhaustion constructive metabolism in most of these cases cannot furnish the amount of restitution which the (normal) function of nerve and cell requires.

Applying the theory to tabes, disturbances of equilibrium and ataxy of gait are the earliest symptoms of this disease, as those nervous apparatus which contribute the muscular sensations necessary for the maintenance of equilibrium and gait are used in an uncommonly high measure. The said symptoms, indeed, are the earliest and most important ones. We also find tabes chiefly in persons who over-exert their legs (military officers, railroad employees, foresters, etc.), while we find it seldom in women who have more sedentary habits. This also explains the rare occurrence of tabes in puellæ publicæ, although they are so frequently affected with syphilis, the indirect etiological importance of which Edinger does not underrate.

The frequency of certain accompanying symptoms, as pupillary disturbances, atrophy of the tongue, of the peroneal muscles, muscles of larynx, etc., is also explained by the unusual amount of function performed by these parts. It is not strange, either, that ophthalmoplegia progressiva is frequently combined with degenerations in the posterior columns.

Edinger considers that the progressive character of progressive general paralysis is also satisfactorily explained by his theory. He is convinced that the nerve tissue is always first affected in this disease. The more nervous substance that is destroyed the more do the remaining healthy parts of it become exerted, which again causes further decay of nervous tissue, etc.

The numerous forcible arguments put forth in defence of Edinger's theory speak highly for its validity. Its application to the comprehension of tabes in its manifold varieties and of the progressive character of general progressive paralysis, seems particularly convincing. ONUF.

CLINICAL.

The Differential Diagnosis of Encephalasthenia.—By Dr. Julius Althaus (*Deutsche Zeitschrift für Nervenheilkunde*, Vol. v., part 6).

The diagnosis of encephalasthenia, often a very easy one, leads some times to fatal mistakes. It is especially liable to be confounded with one of the following: Cerebral tumors and general paresis in early stages of somewhat latent or irregular course; thrombotic softening of the brain, hysteria, hypochrondriasis, and uric acid diathesis (lithæmia).

The absence of constant objective symptoms in encephalasthenia is principally responsible for the difficulty of diagnosis we are often confronted with in dealing with cases of this kind. According to the theory of the author that there are anatomically separated centres in the medulla for different vegetative functions, as, for instance, centre for the heart action, for the action of the glands, for the action of the kidneys, etc., isolated or combined functional disturbances are liable to occur in each of them, and produce clinical symptoms. Some of the latter show so much frequency and constancy as to justify their diagnostic importance, and prove of great value in comparison with the general limitation of objective symptoms in encephalasthenia.

Low specific gravity of the urine (1004-1002), neutral or alkaline reaction, and excess of phosphates are often met met with in encephalasthenics. Polyuria, slight degree of glycosuria (over 0.50-100) and constant or intermittent albuminuria are not very rare phenomena. Oxaluria is very rare, and by no means the cause of the encephalasthenic symptoms, as Prout believes. The careful examination of the urine in every "nervous" patient will help to avoid mistakes of diagnosis. Antipyrin has proved to be of nearly specific therapeutic power in the treatment of nervous (encephalasthenic) glycosuria.

Exaggeration of the tendon reflexes, especially the patellar reflexes, without muscular rigididity, though not constant, is a common occurrence in encephalasthenics.

Lastly, the absence of symptoms known to be produced by an anatomical lesion of the central or peripheral nervous system is in most of the cases conclusive.

The subjective symptoms of encephalasthenia are familiar; from the standpoint of a differential diagnosis the various morbid fears and paræsthesias of the head will sometimes be of great help.

Cerebral hæmorrhage and embolic softening of the brain seldom need be taken into consideration in the diagnostic raisonnement.